

# Inbreeding, heterozygote advantage and the evolution of neo-X and neo-Y sex chromosomes

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Associations between heterozygosities at different loci are generated by inbreeding. This can cause a fusion or translocation involving a sex chromosome and an autosome to have a selective advantage, when there is selection in favour of heterozygotes. Population genetic models of Y-autosome and X-autosome rearrangments in populations mating by a mixture of full sib-matings and random mating are described, in which the rearrangements cause an autosomal locus with heterozygote advantage to become linked to the true sex chromosomes. Such rearrangements gain a selective advantage under a wide range of conditions. If they can invade, Y-autosome rearrangements always spread to fixation, whereas X-autosome rearrangements may be maintained as stable polymorphisms. The results are discussed in relation to data on breeding systems and karyotypic evolution in termites.

**Keywords:** sex chromosomes; translocations; termites; inbreeding; heterozygote advantage

#### 1. INTRODUCTION

In some groups of animals and plants, reciprocal translocations or centric fusions between the sex chromosomes and autosomes have led to the evolution of neo-sex chromosome systems, in which one or more autosomes cosegregate with the sex chromosomes (Westergaard 1958; White 1973; Barlow & Wiens 1976; Fontana 1990; Luykx 1990). The evolutionary causes of such systems raise an intriguing question. Three main answers to this question can be envisaged, which avoid ad hoc assumptions such as a fortuitous association between a rearrangement that creates a neo-sex chromosome system and a mutation with a selective advantage.

The first postulates that genetic drift causes an X-auto-some or Y-autosome fusion or reciprocal translocation to spread through the population, replacing the ancestral karyotype (Charlesworth *et al.* 1987). The second possibility is that one or more loci are maintained polymorphic by selection, with sexually antagonistic effects on fitness such that some alleles are favoured in males and others in females. This will lead to an advantage to an X-A or Y-A translocation or fusion that brings the selected locus or loci into proximity with the sex determining factor (Charlesworth & Charlesworth 1980). Evidence concerning these possibilities is discussed by Charlesworth & Charlesworth (1980), Charlesworth *et al.* (1987) and Rice (1987, 1998).

The third possibility has previously been discussed in relation to the evolution of permanent heterozygosity for reciprocal translocations in highly selfing populations of plants (Lewis & John 1963; De Waal Malefijt & Charlesworth 1979). In inbreeding populations, associations are generated between heterozygosities at different loci (Haldane 1949; Cockerham & Weir 1968). A translocation or fusion heterozygote often reduces the frequency of

crossing over within parts of the chromosomes involved (Dobzhansky 1931; Lewis & John 1963; Davisson & Akeson 1993); heterozygosity for a translocation thus acts as a marker for heterozygosity for some of the genome. If sufficient heterosis is present, this can lead to the spread and maintenance of a rearrangement. Models of selection for reduced recombination among multiple loci maintained polymorphic by heterozygote advantage have been studied by Feldman & Balkau (1972) and Charlesworth et al. (1977), and validate this intuition. Models of heterosis caused by recurrent deleterious mutations suggest, however, that an unrealistically high mutation rate is required to overcome the rapid loss of translocation heterozygosity in a highly inbreeding population (De Waal Malefijt & Charlesworth 1979). Mutational heterosis is therefore likely to be, at best, merely a contributory factor in the establishment of autosomal rearrangements.

But a sex-chromosome/autosome rearrangement does not suffer such a severe problem of loss of heterozygosity under close inbreeding. With male heterogamety, a Y-A fusion or reciprocal translocation is always present in males, so that the autosome attached to the Y never becomes homozygous by descent in the absence of crossing over. An X-autosome rearrangement can become homozygous in females, but is never homozygous by descent in males. There is therefore much greater scope for the maintenance of an association between heterozygosity for the rearrangement and heterozygosity for alleles affecting fitness than in the case of purely autosomal rearrangements. The existence of heterosis would thus be expected to be much more likely to create a selective advantage to a rearrangement. Consistent with this effect, complex systems of neo-X and neo-Y chromosomes have evolved repeatedly in some highly inbreeding species of animals, notably the lower termites (Fontana 1990; Luykx 1990).

Table 1. Offspring arrays for progenies of all possible mating types with a Y-autosome rearrangement

mating types	offspring arrays				
standard genotypes	progeny of each sex				
	$A_1A_1$	$A_1A_2$	$A_2A_2$		
$1A_1A_1 \times A_1A_1$	1	0	0		
$2\vec{A}_1\vec{A}_1 \times \vec{A}_1\vec{A}_2$	0.5	0.5	0		
$3A_1A_1 \times A_2A_2$	0	1	0		
$4 A_1 A_2 \times A_1 A_2$	0.25	0.5	0.25		
$5 A_2 A_2 \times A_1 A_2$	0	0.5	0.5		
$6 A_2 A_2 \times A_2 A_2$	0	0	1		
rearrangement genotypes	female progeny			male progeny	
	$A_1A_1$	$A_1A_2$	$A_2A_2$	$A_1^*A_1$	$A_1^*A_2$
$1 A_1 A_1 \times {A_1}^* A_1$	1	0	0	1	0
$2 A_1 A_2 \times A_1^* A_1$	0.5	0.5	0	0.5	0.5
$3 A_2 A_2 \times A_1^* A_1$	0	1	0	0	1
$4 A_1 A_1 \times A_1^* A_2$	0	1	0	1	0
$5 A_1 A_2 \times A_1^* A_2$	0	0.5	0.5	0.5	0.5
$6 A_2 A_2 \times A_1^* A_2$	0	0	1	0	1

There is therefore a need to investigate quantitatively the question of the nature of selection for neo-X or neo-Y chromosomes in inbreeding populations. The causes of inbreeding depression and heterosis are still a matter for debate, but it seems likely that both deleterious mutations and alleles maintained by heterozygote advantage play a role, with the former probably being more important (Crow 1993; Charlesworth 1998). It is, however, much simpler to model the case of a single locus with heterozygote advantage rather than a multi-locus model of mutation and selection, and this is the subject of the present paper. The results confirm that there is, indeed, often selection for a fusion or translocation between a sex chromosome and an autosome in a partially inbreeding species when there is heterozygote advantage.

## 2. A Y-AUTOSOME TRANSLOCATION OR FUSION

#### (a) The model

We assume that the population is initially in equilibrium with respect to a single autosomal locus with two alleles,  $A_1$  and  $A_2$ . Variation is maintained by heterozygote advantage, such that the homozygotes for  $A_1$  and  $A_2$  have fitnesses 1-s and 1-t, respectively, relative to the fitness of  $A_1A_2$ . The population is assumed to be infinite in size; a fraction of matings,  $\alpha$ , are between full siblings; the remainder take place at random. The fitnesses of genotypes are assumed to be independent of the composition of the family to which they belong. An exact description of the dynamics of the system involves a vector  $\boldsymbol{x}$  of six dimensions, whose elements are the frequencies of matings between all possible pairs of genotypes  $(A_1A_1 \times$  $A_1A_1$ ,  $A_1A_1 \times A_1A_2$ , etc.). The recursion relations for the new vector of frequencies,  $\mathbf{x}'$ , are easily written down from the progeny arrays for each mating type, as can be seen from table 1 and Appendix A.

These equations can be used to generate expressions for equilibria (Page & Hayman 1960; Karlin 1968), but the composition and stability of an equilibrium for a given set of parameters are more easily obtained by iteration of the recursion relations. If there is a very high frequency of inbreeding, and s and t differ sufficiently, there may be no stable polymorphic equilibrium, since the allele associated with higher homozgygous fitness has a net selective advantage (Page & Hayman 1960). If there is 100% sib-mating, and selection is insufficiently strong to counter the decline in heterozygosity caused by inbreeding, the population can be maintained polymorphic, but no heterozygotes are present at equilibrium (Hayman & Mather 1953; Wright 1969, p. 247). This situation is somewhat unrealistic, and would prevent the spread of a rearrangement, since a new rearrangement would carry the same A allele as the other individuals with which it mates. Populations with 100% sib-mating were therefore not studied.

The introduction of a Y-autosome rearrangement was modelled as follows, assuming that recombination is completely suppressed between the centromere of the fused chromosome and the locus under selection. It was arbitrarily assumed that the rearrangement involves a Ychromosome and an  $A_1$ -bearing autosome. There are thus only two possible genotypes that carry the rearrangement, both of which are male:  $\Upsilon$ - $A_1/X$ ;  $A_1$  and  $\Upsilon$ - $A_1/X$ ;  $A_2$ . These will be denoted by  $A_1^*A_1$  and  $A_1^*A_2$ , respectively. There is a total of six types of mating involving these genotypes, whose frequencies form a vector y. Table 1 summarizes the progeny arrays produced by these matings. The resulting recursion relations are given in Appendix A and in the electronic appendix (see the Royal Society Web site at http://www.pubs.royalsoc.ac. uk/publish/pro\_bs/jan99pbl.htm).

## (b) The results

To study the dynamics of the rearrangement, it was introduced into an equilibrium population at a frequency of 0.001, in males of the genotype  ${A_1}^*A_1$ , corresponding to a new chromosomal mutation that arises in a population of moderate size. For convenience, the matings involving this genotype were assumed to occur in random-mating proportions in the first generation. The asymptotic rate of increase in log frequency of the rearrangement when rare

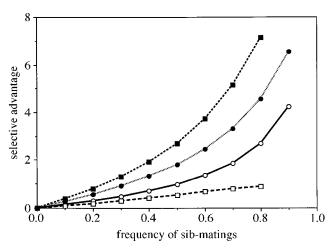


Figure 1. The per cent selective advantage of a rare Y-auto-some rearrangement, plotted against the frequency of sib-matings for several modes of selection: s=t=0.1 (open circles), s=t=0.2 (filled circles), s=0.1, t=0.2 (open squares), s=0.2, t=0.1 (filled squares). Cases where no initial polymorphism are not maintained are not shown.

(less than 1% in frequency) provides a measure of the selective advantage of the rearrangement, k; the probability of fixation of the rearrangement in a large stationary population is ca. 2k (Crow & Kimura 1970, p. 422).

The runs showed that, when there was a stable initial polymorphism at the A locus and selection was not too strong, the rearrangement spread to fixation in all cases. Its initial selective advantage was always very small with 100% random mating, reflecting an algebraic rather than a geometric rate of increase, i.e. k is a decreasing function of its initial frequency,  $p_0$ , so that the rearrangement had a negligible advantage in a very large population. When there was some sib-mating, k did not depend strongly on  $p_0$ , so that the selective advantages displayed in figure 1 are good approximations provided that  $p_0 \ll 1$ .

The results indicate that even a moderate level of inbreeding can induce a significant selective advantage to a new rearrangement; the advantage increases with the level of inbreeding, until heterozygosity ceases to be maintained. The selective advantage is larger with more intense selection; with symmetric selection, the maximum value of k is about one-half s. With asymmetric selection, k is greater when the rearrangement is associated with the less fit allele than when it is associated with the fitter allele. This is at least partially offset by the lower probability that a rearrangement will arise in a gamete carrying the less fit allele, since there is a considerable asymmetry in allele frequencies when inbreeding is high. For  $\alpha = 0.8$ , the products of the frequencies of  $A_1$ and the respective k values are 0.011 for s=0.1, t=0.2, and 0.007 for s = 0.2, t = 0.1.

It was also found that the rearrangement could experience a selective disadvantage when selection was very strong, and the level of sib-mating exceeded a critical value; e.g. with s=t=0.8, there was a disadvantage to the rearrangement when  $\alpha>0.45$  but an advantage with lower rates of sib-mating. There is no obvious intuitive explanation for this disadvantage, which is probably of little biological significance in view of the extreme parameter values required.

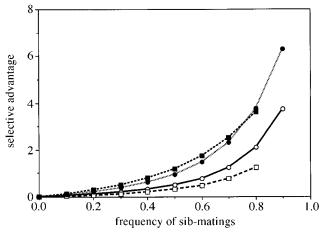


Figure 2. The per cent selective advantage of a rare X-auto-some rearrangement, plotted against the frequency of sib-matings for several modes of selection (symbols as in figure 1).

#### 3. AN X-AUTOSOME TRANSLOCATION OR FUSION

## (a) The model

The selection and mating regimes are identical to the above. The rearrangement is now assumed to be between the X-chromosome and an autosome carrying  $A_1$ , with no recombination between the centromere and  $A_1$ . The rearrangement haplotype is denoted by  $A_1^{**}$ . A 21-dimensional vector z is required to describe the possible matings involving  $A_1^{**}$ . The recursion relations are correspondingly complex and are given in the electronic appendix. The initial selective advantage of the rearrangement, k, was studied in the same way as for a Y-autosome rearrangement. This model applies equally well to a system of X0 sex determination, since the presence of the Y chromosome makes no difference to the dynamics of an X-A rearrangement.

## (b) The results

Figure 2 shows the relation between k and the frequency of sib matings. As before, the existence of a geometric initial advantage is dependent on the occurrence of inbreeding. The selective advantage was generally lower than in the Y-autosome case, as might be expected from the fact that an X-autosome rearrangement can become homozygous in females. This difference was especially marked for low levels of inbreeding, with k values of less than half of those for Y-A rearrangements In addition,  $A_1^{**}$  often did not spread to fixation, but instead generated a balanced polymorphism, except for high levels of inbreeding, especially when s>t. This reflects a heterozygote advantage for the rearrangement, due to the preferential association of  $A_1$  with the rearrangement. As before, with intense selection and high inbreeding, there may be selection against a rearrangement; e.g. when s=t=0.8, there was a disadvantage to the rearrangement when  $\alpha > 0.51$  but an advantage with lower rates of sib-mating.

#### 4. DISCUSSION

The results described above show that heterozygote advantage at a single locus in a population mating by a

mixture of inbreeding and random mating can promote the evolution of reciprocal translocations or fusions between the sex chromosomes and autosomes. This is not possible for purely autosomal rearrangements, which require multiple loci maintained by heterozygote advantage to be favoured (Feldman & Balkau 1972; Charlesworth et al. 1977). For low levels of inbreeding ( $\alpha$ <0.5), there is considerably stronger selection on rare Y-A rearrangements than on X-A rearrangements; the two cases tend to converge for higher levels of inbreeding, except when there is strong selection against homozygosity for the allele associated with the rearrangement. Polymorphism for X-X rearrangements is also possible, but Y-A rearrangements always become fixed. We might therefore expect that (a) there should be an association between partial inbreeding and the evolution of neo-sex chromosome systems; (b) that Y-A rearrangements would be more frequently established and should be fixed within populations; and (c) polymorphism for X-A rearrangements within populations should sometimes be observed.

Tests of these predictions are hard to conduct, primarily because of the lack of reliable data on factors such as levels of inbreeding within populations and withinpopulation variation in karyotypes. In addition, there may be confounding effects of other factors. For instance, the division of a population into a set of small, isolated colonies, with rapid turnover due to local extinction and recolonization, may cause inbreeding effects of the kind described here, but will also favour the establishment by drift of rearrangements with heterozygote disadvantage (Wright 1941; Lande 1985). Nevertheless, the high rate of evolution of new neo-sex chromosome systems in lower termites, often involving multiple rearrangements (Fontana 1980, 1990; Luykx 1987, 1990; Luykx & Syren 1981; Syren & Luykx 1977, 1981), seems to fit the present model better than the others.

In Luykx's (1990) study of 25 species of lower termites from Australia, the rate of establishment of new X-A or Y-A rearrangments was three times greater than the rate for A-A rearrangements, so that the effect seems to be more than a mere acceleration of the rate of evolution of rearrangements in general. Such an excess could in principle be explained by the drift and sexual antagonism models (Charlesworth & Charlesworth 1980; Charlesworth et al. 1987), but the latter provides no explanation for why it is specifically lower termites that preferentially acquire rearrangements involving the sex chromosomes. The very high rate of incorporation of rearrangements suggests that they must be close to neutral if the drift hypothesis is correct, in which case it is difficult (without further ad hoc assumptions) to account for the higher rate of sex chromosome rearrangements compared with autosomal ones.

An excess of Y-A rearrangements is expected on both the present model and on the sexual antagonism model (Charlesworth & Charlesworth 1980), as are the observed associations of allozyme marker alleles with the neo-sex chromosomes (Luykx 1981; Santos & Luykx 1985). In the Australian lower termites, there are five Y-A versus one X-A rearrangements among cases where the nature of the sex chromosome could be determined (Luykx 1990); p=0.03, assuming that X-A rearrangements are three times as likely as Y-A rearrangements. Note, however, that Luykx & Syren (1983) inferred the occurrence of four X-A rearrangements and only one Y-A rearrangement in Kalotermes approximatus. Polymorphisms for neo-sex chromosomal rearrangements have been observed in termites (Fontana 1980; Syren & Luykx 1981); whether these involve X-A as opposed to Y-A rearrangements is unknown, and the possibility that they merely reflect hybrids between formerly isolated populations cannot be excluded.

In lower termites, non-reproductive individuals may become reproductively active following the deaths of colony founders, and then mate with individuals from the same colony (Fontana 1990; Thorne 1997). This leads to inbreeding, although matings between winged individuals from different colonies probably ensures some degree of outbreeding as well (Thorne 1997). In *Incisitermes schwarzi*, which possesses a complex system of neo-sex chromosomes (Syren & Luykx 1977), ca. 40% of colonies have replacement reproductives from within the colony (Luykx 1993a), implying a high level of inbreeding. Higher termites have more stable karyotypes than lower termites (Luykx 1990); it remains to be established if this reflects less frequent replacement of reproductives in higher termites (with their sterile castes), and hence lower inbreeding (Myles & Nutting 1988). More comparative studies to test whether there is truly an association between inbreeding and the evolution of neo-sex chromosomes are clearly desirable.

Some limitations of the model should be noted. First, we have assumed no recombination between the centromere of the rearrangement chromosome and the locus subject to selection. This is justified by the fact that, as mentioned in § 1, recombination is often greatly reduced in frequency in translocation heterozygotes; the frequency of recombination between two allozyme loci and the centromere of the neo-sex chromosomes of Incisitermes schwarzi is of the order of 0.5% (Santos & Luykx 1985), whereas two autosomal allozyme loci show free recombination (Luykx 1993b). Given previous results (Charlesworth & Charlesworth 1980), we would expect recombination to diminish but not abolish the selective advantages calculated here. It is unclear whether the conclusion that polymorphism is possible for X-A rearrangements is robust in the presence of recombination. Second, we have assumed that heterosis is caused by a single locus with heterozygote advantage. Since homozygosity for a chromosome segment in an inbreeding population is likely to be associated with reduced fitness in much the same way as homozygosity for an allele at a locus with heterozygote advantage, the present study probably provides a rough guide to what is likely to be true in the case of mutational heterosis (Charlesworth 1991). A detailed investigation of this case by computer simulations is needed to verify this conjecture.

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## APPENDIX A. RECURSION RELATIONS FOR A Y-AUTOSOME REARRANGEMENT

A vector  $\mathbf{x}$  is used to represent the frequencies of the set of six matings involving standard genotype males listed in table 1, and a vector  $\mathbf{v}$  to represent the frequencies of the six matings involving rearrangement males. The mean fitnesses of female and male progeny are given by the following expressions:

$$\overline{w}_{f} = (x_{1} + y_{1})(1 - s) + (x_{2} + y_{2})(1 - 1/2s) + x_{3} + y_{3} + x_{4}(1 - 1/4[s + t]) + y_{4} + (x_{5} + y_{5})(1 - 1/2t) + (x_{6} + y_{6})(1 - t)$$
(A1a)

$$\begin{split} \overline{w}_{\rm m} &= (x_1 + y_1)(1-s) + (x_2 + y_2)(1-1/2s) \\ &+ x_3 + y_3 + x_4(1-1/4[s+t]) + y_4(1-s) \\ &+ x_5(1-1/2t) + y_5(1-1/2s) + x_6(1-t) + y_6 \end{split} \tag{A1b}$$

The eight-dimensional row vector  $\boldsymbol{u}$  represents the zygotic frequencies of the genotypes of females and males; the first three elements of  $\boldsymbol{u}$  are the zygotic frequencies of  $A_1A_1$ ,  $A_1A_2$  and  $A_2A_2$  females, respectively; the next three elements are the zygotic frequencies of  $A_1A_1$ ,  $A_1A_2$  and  $A_2A_2$  males; the last two are the zygotic frequencies of  $A_1^*A_1$  and  $A_1^*A_2$  males. Let the 12-dimensional row vector v be the composite vector  $(\mathbf{x}, \mathbf{y})$ . Let the  $12 \times 8$  matrix G be formed by combining the matrices of progeny frequencies generated from each mating type, with rows corresponding to mating type and columns to the elements of  $\mathbf{u}$ . The new set of zygotic frequencies is then given by:

$$\mathbf{u}' = \mathbf{v} G \tag{A2}$$

The post-selection frequencies of the genotypes of females and males are given by multiplying the elements of  $\mathbf{u}'$  by the fitnesses of the corresponding genotypes, normalized by the mean fitnesses of females or males from equations (Al), as appropriate; these frequencies form another eight-dimensional vector  $\boldsymbol{p}$ . The contribution from random mating to the new frequencies of the various mating types is obtained by multiplying the appropriate products of the components of p by  $1 - \alpha$ . The contribution from sib-mating is obtained by assuming random mating among the surviving progeny of each type of mating in table 1. These assumptions yield a set of 12 recurrence relations, given in the electronic appendix.

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